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Fat Embolism Syndrome And Crush Syndrome

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❖ Introduction ❖

Fat Embolism syndrome is a major cause of morbidity and mortality after multiple long bone fractures and is an important cause of ARDS.

FES may be defined as, "a complex alteration of homeostasis that occurs as an infrequent complication of fractures of long bones and pelvis and manifests clinically as acute respiratory insufficiency".

FES develops when fat emboli become impacted in pulmonary microcirculation and other microvascular beds such as the brain and is characterized by respiratory failure, cerebral dysfunction and petechiae. In-patients with pre existing pulmonary disease the addition of FES can be life threatening. FES is an a important cause of acute respiratory distress syndrome. With prompt recognition, the treatment of the fat embolism syndrome has become more specific and less empiric, resulting in decrease morbidity and mortality. In recent years prevention of fat embolism syndrome by early fracture fixation and patient mobilization has become the focus of a wave of clinical investigation.

Acute respiratory insufficiency after skeletal trauma has multiple causes :

- Fat embolism
- Aspiration of gastric contents
- Pulmonary edema
- Airway obstruction
- Pneumonia
- Pulmonary contusions
- Shock lung

❖ Causes ❖

FES occurs most commonly as an early complication of fractures of the pelvis and long bones. FES is also reported in other entities:

- As a complication of reaming of medullary canals of long bones
- As a complication of reaming and cementation during joint replacement ? Massive soft tissue injury ? Severe burns
- Liposuction
- Chronic osteomyelitis
- Metabolic disorders
- Neoplasms
- Renal transplant
- Bone infarcts in hemoglobinopathoies
- Collagen disease
- Diabetes
- Severe infection
- Inhalation anesthesia
- Blood transfusion

❖ Historical Aspects ❖

FES is a well-known entity as a complication of long bone fractures from one and half centuries.

Zenker in 1861 described fat droplets in the lung capillaries of a rail-road worker with fatal thoraco-abdominal crush injury.

Wagner in 1865 described the pathologic features of fat embolism.

Von Bergmann in 1873 was the first to establish FES in a patient with femoral fractures by postmortem demonstration of a large amount of pulmonary fat.

Czerny in 1875 described the symptoms of FES and the importance of fundoscopic examination in its diagnosis.

Fenger and Salisbury in 1879 made the first clinical diagnosis of FES in a patient with femoral fractures and confirmed it by demonstrating massive fat emboli in the lung at autopsy.

❖ Incidence ❖

The exact incidence of FES is not known. The clinical signs and symptoms develop in 0.5% to 2% of patients with long bone fractures and in 10% of patients with multiple fractures. The risk of FES increases with the increased incidence of multiple fractures in major automobile accidents. FES is rare in children probably because of low fat content in their marrow.

❖ Pathogenesis And Pathophysiology ❖

This is a subject of conjecture and controversy. The source of embolic fat is thought by most to be the bone marrow. Bone marrow elements have been demonstrated in lung sections, indicating that mechanical fat embolization does occur. Two theories have been offered for the pathogenesis of FES:

(a) Mechanical theory : This classic theory postulates that triglyceride particles from injured fat marrow enter the circulation and obstruct the pulmonary micro vessels. Fat globules vary in size from 2 to 200 microns and most get lodged in vessels less than 75 microns in diameter.

❖ **The blockage of pulmonary capillaries results in :**

- Venoarterial shunting
- Hypoxemia and
- Alveolar hypoperfusion leading to pulmonary dysfunction.

(B) Biochemical Theory : As FES has also been documented in non traumatic disorders, the mechanical theory cannot adequately explain the phenomenon. Alternative or additional mechanisms have therefore been suggested. Peltier, Barie and others proposed a biochemical theory which incriminates free fatty acids.

According to them, local hydrolysis of triglycerides and neutral fat emboli by pneumocyte lipase results in increased free fatty acids.

❖ **Free fatty acids have been shown to be toxic to lung parenchyma and lead to**

- Disruption of alveolar capillary membrane
- Decreased surfactant production
- Interstitial hemorrhage and
- Pulmonary edema resulting in pulmonary dysfunction.

Thus, the latent period before clinical manifestations appear may be explained by the time needed for lipoprotein lipase to convert neutral fat to toxic free fatty acids.

❖ Clinical Presentation ❖

Onset of symptoms is usually within 12 to 72 hours but may manifest as early as 6 hours and as late as 10 days.

Arterial hypoxemia is the hallmark of FES and the clinical manifestations are a result of reduced blood flow to the lungs and brain

Early symptoms are :

- Shortness of breath
- Restlessness
- Confusion

The classic triad of FES involves :

- Pulmonary dysfunction
- Cerebral dysfunction and
- Cutaneous changes.

Pulmonary Dysfunction

Features of pulmonary insufficiency are the earliest signs of FES and include :

- Tachypnoea with respiratory rate more than 30 per minute
- Dyspnoea
- Cyanosis

- Rales and rhonchi
- Respiratory failure and ARDS
- Occasionally hemoptysis and pulmonary edema

Cerebral Dysfunction

Features include :

- Restlessness
- Confusion and disorientation
- Irritability and delirium
- Stupor and coma
- Convulsions
- Diffuse neurological deficit.

The neurological features in FES appear to change periodically and may sometimes progress very rapidly.

Cutaneous Changes

Are characterized by :

- Petechiael rashes located in the upper anterior chest, axilla, neck, oral mucous membrane and conjunctiva.
- Appear on the second to third day
- May occur periodically with accompanying attacks of coma
- Resolve within 7 days

The distribution of petechiae is theorized to the related fat particles floating in the aortic arch and embolising to non-depended skin areas via subclavian and carotid arteries.

The other signs of FES include :

- Pyrexia : Fracture fever / haematoma fever
- Tachycardia: pulse rate more than 140 per minute
- Retinal changes which include edema, hemorrhage or intravascular fat globules as seen on fundoscopy
- Renal changes like lipuria
- Hepatic changes like jaundice
- Urinary incontinence

Sevitt classified FES into 3 distinguishable clinical presentations :

- **Sub clinical FES**
- **Non fulminant FES**
- **Fulminant FES**

Sub clinical FES

- Probably occurs in almost all long bone fractures of the lower extremity and fractures of the pelvis
- Characterised by decreased PaO2, decreased Hb% and decreased platelets. No clinical signs and symptoms of respiratory insufficiency.

Non-fulminant FES

- Clinical signs and symptoms are clearly evident.
- Respiratory insufficiency, cerebral symptoms and petechiae appear classically
- Typical radiological and haematological changes can be detected

Fulminant FES

- Rarer form and appears within hours of injury
- Characterised by severe respiratory failure and altered mental status and convulsions.

FES should be Strongly Suspected if the Patient has

- Unexplained Dyspnoea and Tachycardia
- Unexplained confusion and cerebral dysfunction
- Petechiae in the upper half of the body.

Often the signs and symptoms may be masked by shock, coma, head injury and anesthetic drugs.

❖ Diagnosis ❖

The diagnosis of FES is essentially by clinical features and there are no pathognomonic tests for confirmation. However certain laboratory and x-ray features aid in the diagnosis of FES.

Laboratory Investigations

- Sustained reduction in PaO2 levels (partial

pressure of oxygen in arterial blood) below 60 mm of Hg as detected by arterial blood gas analysis indicates FES. Serial determination of PaO2 levels is necessary. Clinical features appear only when the levels fall below 65 mm of Hg.

- Thrombocytopenia; platelets below 150000 cell per cubic millimeter is commonly seen.
- Detection of fat globules in urine, sputum, CSF and blood; usually difficult to do. Gurd suggested detection of pathological fat in filtered venous blood. Cryostat frozen section of clotted blood may reveal fat globules.
- Biopsy of petechiael skin lesions may reveal presence of fat.
- Elevated serum lipase and free fatty acid levels
- Broncho alveolar lavage and detection of fat droplets within cells recovered by lavage may aid in rapid diagnosis of FES.

❖ ECG ❖

Show only non-specific changes like T wave inversion, prominent S waves and occasional arrhythmias.

X-ray

Chest X-ray reveals a diffuse fluffy bilateral infiltrate classically called "snow storm" appearance. This may progress to widespread airspace consolidation caused by alveolar hemorrhage and edema.

A differential diagnosis of cardiogenic edema and traumatic lung contusion must be kept in mind.

Since there are no definitive and pathognomonic features of FES, several authors have suggested some aids in diagnosing FES.

Gurd and Wilson have divided the features of FES into major and minor criteria.

❖ Gurds Criteria ❖

Major Features (at least one)	Minor Features (at least four)
◆ Respiratory insufficiency	◆ Pyrexia
◆ Cerebral dysfunction	◆ Tachycardia
◆ Petechiael rash	◆ Retinal changes
	◆ Jaundice
	◆ Renal changes

Laboratory Features (at least one)
◆ Fat macroglobulinemia
◆ Anemia
◆ Thrombocytopenia
◆ High ESR

They proposed that if any one major and four minor features are present after a latent period after injury, then a diagnosis of FES could be made.

Schonfeld et al proposed a quantitative means of diagnosing FES

❖ Schonfeld's Fat Embolism Index ❖

Symptoms	Score
◆ Petechiae	5
◆ Diffuse alveolar infiltrates	4
◆ Hypoxemia (PaO2 less than 9.3 kPa)	3
◆ Confusion	1
◆ Fever (more than 38'C)	1
◆ Tachycardia (more than 120 beats per min)	1
◆ Tachypnea (more than 30 per min)	1

A cumulative score greater than 5 is necessary for a positive diagnosis of FES.

❖ Differential Diagnosis ❖

Since FES involves respiratory insufficiency and cerebral dysfunctions other conditions causing the same features should be kept in mind.

The Respiratory Insufficiency Should be Differentiated From

- Pulmonary contusion
- Pulmonary embolism
- Bronchial pneumonia

Radiological Features Differentiating FES From Traumatic Lung Contusion are

Features	FES	Lung Contusion
◆ Onset of X-ray changes after trauma.	1 to 2 days	Immediate
◆ Distribution	Bilaterally symmetrical	Unilateral or asymmetrical

Cerebral Dysfunction Should be Differentiated From

- Subdural or extradural haematoma
- Cerebral contusion.
- Diabetic coma

Clinical Features Differentiating Cerebral Fat Embolism From Craniocerebral Trauma

Symptoms and Signs	Cerebral FES	Craniocerebral Trauma
◆ Lucid interval	48 to 72 hours	6 to 10 hours
◆ Confusion	Severe	Moderate
◆ Pulse rate	Rapid more than 140/min	Slow
◆ Onset of coma	Rapid	Slow
◆ Localising signs	Usually absent	Usually present
◆ Decerebrate rigidity	Early	Terminal

❖ Treatment ❖

The treatment of FES consists of general measures and specific measures

General Measures

Include

- Maintenance of airway.
- Maintenance of fluid and electrolyte balance.
- Blood transfusion.
- Immobilization of fractures in splints / POP to prevent further emboilization ?J* Analgesics and when indicated antibiotics.
- Monitoring of BP, urinary output, PaO2.

Specific Measures

Respiratory Support is the Mandatory in the Management of FES

- Oxygen therapy via mask / nasal cannula to maintain PaO2 at 90 mm of Hg ?> Endotracheal intubation and mechanical ventilation if respiratory distress is impending
- Corticosteroids; methyl prednesolone 10 mgs/kg/daily in 3 divided doses i.v. should be administered. It helps by;
  - Inhibiting the inflammatory response of the lungs to emboli
  - Limiting the decrease in PaO2

- Ethanol / alcohol: accidental evidence shows those intoxicated patients fare better after multiple fractures and FES. It has been suggested that a bolus of alcohol may prevent and limit the effects of FES probably by acting as a lipase inhibitor.
- Other agents like heparin and hypertonic glucose etc. have been tried with no measurable success.

Fracture Stabilization

It has been shown by many authors that early fracture fixation by unreamed intramedullary nailing considerably reduces the incidence of FES. Early internal fixation of fractures aids by,

- Allows the patient to be upright and thus improves lung function
- Decompresses the fracture haematoma and thus eliminates source of emboli
- Eliminates pain and stress of motion at fracture site and reduces need for opiate analgesics

Prophylaxis of FES

We have all been taught that prevention is better than cure. The various measures we can take to prevent FES in an injured patient are,

- Gentle handling, proper splinting and careful transport of the patient with multiple injuries.
- Aggressive treatment of hypovolemic shock
- Early stabilization of fractures
- Reduce bone marrow release into circulation during preparation of medullary canal for intramedullary nailing / prosthesis insertion.
- Identification of patients at risk by regular monitoring with pulseoximetry, blood gas analysis etc.

❖ Prognosis ❖

FES in its subclinical form is self-limiting and recovery is almost complete. FES in its non-fulminant and fulminant forms needs early recognition and aggressive treatment for the patient to recover. Mortality is still close to 5% to 15% and is closely related to severity of respiratory dysfunction. Morbidity is usually secondary to focal cerebral neurological deficit.

Intramedullary Reaming and FES

- Reaming of the medullary canal prior to insertion of a nail or a prosthesis is know to increase the incidence of FES.
- Insertion of femoral stem during hip replacement increases the intramedullary pressure and may lead to embolisation of fat particles and bone marrow leading to FES.
- In these instances not only fat particles but other substances like tissue particles, thromboplastin, fibrin degradation products etc may be the culprits
- It is therefore wise to use unreamed nails in patients with multiple injuries.

❖ Summary ❖

- Fat embolism, usually sub clinical occurs in the majority of patients with fractures of pelvis and long bones.
- Respiratory insufficiency, cerebral dysfunction and petechiae appearing 48-72 hrs after multiple fractures are considered pathgnomonic of fat embolism.
- Despite certain laboratory and radiological diagnostic aids, clinical features are still the corner stone for diagnosis of fat embolism.
- Treatment consists of respiratory support, volume replacement, cortico-steroid

therapy and possible fracture fixation.

- Early detection, careful monitoring and aggressive therapy have considerably reduced the morbidity and mortality due to fat embolism.
- In spite of all available therapy, fulminant fat embolism with severe respirator}' failure can still be fatal.

❖ Crush Syndrome ❖

The term crush syndrome, or traumatic rhabdomyolysis, refers to the sequela of prolonged continuous pressure on muscle tissue. Crush syndrome refers to the systemic manifestation associated with crush injuries, such as hyperkalemia, Myoglobinemia and anuric renal failure.

❖ Causes ❖

Commonly Seen in

- Earth quake victims
- Bombings
- Train accidents
- Motor vehicle accidents
- Prolonged application of military anti shock trousers

❖ Pathophisiology ❖

Increased intramuscular pressure - more than 240 mm / hg

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Muscle break down (direct pressure)

↓

Ischemia and necrosis of muscles (independently)

↓

Reperfusion of ischemic necrotic muscle with oxygenated blood

↓

Reperfusion myopathy (second insult)

↓

Forms reactive oxygen metabolites.

↓

Xanthine oxidase + hypoxanthine + molecular oxygen releases in skeletal muscle

↓

Rapid production of super oxide radicals and hydrogen peroxide

↓

Failure of ion pumps and increased membrane permeability of myocytes and micro vasculature

↓

Fluid shift produces shock (extensive amount of muscle involved)

↓

Release of potassium, phosphorus, lactic acid and myoglobin circulation

↓

Cardiac arrest, shock, acidosis and renal failure.

❖ Clinical Features ❖

Local Features

- Edema with or without cellulitis
- Hematoam with or without abscess
- Copartment syndrome due to extensive muscle edema

Systemic Complication

- Hypovalemia
- Anemia
- Hypotention
- Tachycardia
- Electrolyte abnormalities like .
  - Hyperkalemia
  - Hypocalcemia
  - Phosphorus and magnesium imbalance
  - Coagulopathy
  - Renal failure (5-15%)

❖ Investigation ❖

- Hematocrit
- K,Na,Ca,P
- Platelet Count
- Blood urea
- Serum creatanine
- CPK
- Urine for pigment
- Blood gases
- Appropriate radiographs

❖ Treatment ❖

The treatment of crush syndrome is divided in to systemic treatment and local treatment.

Systemic Treatment

Begins with in an hour or immediately after rescue. Treatment should begin immediately by anticipating the onset of this syndrome.

- Aggressive fluid resuscitation is the mainstay of treatment.

  1500 ml / hour crystalloid saline infusion in adults to maintain a urine out put of at least 100 ml / hour.

- Administer 1 gr/k.g. Mannitol and 100 mEq bicarbonate in order to increase diuresis, which interns increases the excretion of urine and prevents renal failure.

- Administration of Allopurinol limits reperfusion by inhibiting xanthine oxidase activity.

- Correct electrolyte imbalance.

- Renal dialysis required if anuria persist

Local Treatment

The optimal approach to the local injury is still a matter of debate. Most authors recommended conservative management unless until there is threatening compartment syndrome.

Fasciotomy is the treatment of choice In case of severe crush injury wound debridement and exploration can be done.

Reference

Rock wood and green

Pubmed

Medline

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